THE POTENTIAL ROLE OF FLEXOR HALLUCIS LONGUS STENOSIS IN THE PATHOGENESIS OF HALLUX RIGIDUS: A DYNAMIC CADAVER MODEL

+*Kirane, Y M; **Michelson J D; *Sharkey N A
+The Pennsylvania State University, University Park, PA
ymk107@psu.edu

INTRODUCTION

Pathologies of the flexor hallucis longus (FHL), the major great toe flexor, include tendinitis, tenosing tenosynovitis, partial tears, triggering and frank rupture. Many of these conditions involve disruption of normal functional anatomy at the fibro-osseous tunnel behind the medial malleolus. A better understanding of normal and abnormal FHL mechanics at this site may provide further insight into various hallux disorders, enabling better diagnosis and treatment. The goal of this work was to examine the biomechanical consequences of proximal tenosynovitis and stenosis of the FHL. We hypothesized that disruption of normal musculotendinous positioning would increase the forces generated in the FHL tendon and thereby increase axial loading of the first metatarsal through the metatarso-phalangeal (MTP) joint.

METHODS

Six non-embalmed, lower extremities were tested in a loading apparatus (called the RDAS) that re-creates the stance phase of walking (Sharkey and Hamel, 1998). Prior to each experiment, three 350Da strain gauges were mounted on the dorsal, lateral and plantar surfaces of the first metatarsal with cyanoacrylate. All extrinsic foot and ankle tendons were stripped of muscle tissue and linked at their myotendinous junctions via cables and cryogenic clamps to a set of linear actuators incorporated into the RDAS. The actuators simulated the temporal contractile activity of the major extrinsic muscles of the foot using force profiles derived from electromyographic and anthropometric data available in the literature (Perry 1992 and Wickiewicz et al 1983). Another set of actuators simultaneously drove the lower extremity in the sagittal plane using kinematic data taken from live subjects with foot sizes comparable to those of each specimen.

Experimental trials were conducted under different conditions while recording tendon forces and displacements, and first metatarsal strains. During the initial trials with the RDAS operating under force feedback control (FFB), target muscle force profiles were prescribed to all the clamp-tendon units (CTUs) as input while their excursions and the actual generated forces were recorded. Subsequent trials with the FHL under displacement feedback control (DFB) used positional values based upon excursions measured in the original FFB trials to control the FHL-CTU. DFB trials were first run with the FHL-CTU held in its normal neutral position. Subsequent trials were executed with the FHL-CTU held in positions proximally displaced from neutral by 2, 4 and 6 mm. The approach was designed to simulate proximal FHL pathology, specifically stenosing tenosynovitis with progressive fibrosis, adhesion, and inflammation at the fibro-osseous tunnel. All other extrinsic muscles remained under FFB during the entire experiment.

Following the gait simulation trials, the first metatarsal was resected with the strain gauges intact and known axial loads (AX) and bending moments were applied to the bone while recording strain in order to determine calibration coefficients. Subsequently, in-situ metatarsal forces and moments under the various conditions, namely FFB, and DFB with FHL-CTU held in its normal neutral position or in displaced positions, were derived using these coefficients (Carter et al). Recorded variables were averaged across two trials for each condition, and analyzed using ANOVA. Force-time integrals over the stance phase were calculated for FHL tendon force and 1st metatarsal axial force and thereby increased axial loading of the first metatarsal through the metatarso-phalangeal (MTP) joint.

RESULTS

The range of excursion for the FHL-CTU under FFB was 6.14 ± 2.45 mm. Interestingly, holding the FHL-CTU at a fixed position around the middle of its excursion range (neutral position) resulted in force profiles comparable to the target profiles derived from EMG data (Fig 1). This neutral position was specific and unique to each specimen. Progressive proximal positioning by 2 mm increments resulted in corresponding and significant increases in FHL tendon forces (p < 0.001) and axial forces within the 1st metatarsal (Fig 1; p < 0.001). Regressions of tendon to bone force-time integrals demonstrated significant inter-relationships (R² = 47.7 %; p < 0.001).

DISCUSSION

We found that the changing magnitude of contractile stimuli (integrated EMG signal as measured in normal subjects) to the FHL over the stance phase of gait corresponds almost exactly to the magnitude of forces experienced at the myotendinous junction when that junction is held in a constant position. This finding, coupled with the observed narrow range of FHL excursion when modeling under force control, leads us to conclude that the FHL fiber function isometrically during the stance phase of normal gait. It has been previously postulated that many multi-articular muscles act isometrically to transfer mechanical energy from one joint to another with minimal change in muscle fiber length (Bobbert and van Ingen Schenau 1987). Our findings strongly suggest that the FHL operates in this fashion, using a neural drive that serves to maintain a specific muscle fiber length rather than a force. In this scenario, the changing magnitudes of motor stimuli (EMG) occur in response to angular displacements at the ankle and 1st MTP joints that would tend to lengthen the entire musculo-tendinous unit. This is most apparent in the early push off phase when both the 1st MTP joint and ankle are dorsiflexed.

Normally the distal muscle fibers and myotendinous junction of the FHL reside in the funnel-shaped entrance to the fibro-osseous tunnel, more distally the tunnel is fused with the periosteum of the calcaneus and talus. Thus, it can be argued that with a narrow or isometric operational range, gliding of the FHL tendon in the fibro-osseous tunnel during normal walking mainly occurs as a result of the relative motion of the tunnel and the bones against the tendon, rather than vice versa.

Loss of normal gliding has been postulated in various dysfunctional disorders of the FHL, especially, stenosing tenosynovitis, which involves progressive inflammation, fibrosis and adhesion of the FHL tendon inside the fibro-osseous tunnel. Furthermore, a causative relation between FHL pathology and hallux rigidus has been hypothesized recently (Michelson and Dunn, 2005). In the present model, proximal displacement of the FHL-CTU, which simulates progressive swelling and stenosis of the FHL, resulted in significantly higher axial forces within the 1st metatarsal. This study thus, provides corroborative evidence that higher forces can be transmitted through the 1st MTP joint to the 1st metatarsal as a result of proximal inflammation and tenosynovitis. Chronically elevated axial forces across the 1st MTP joint could then be responsible for the degenerative changes seen in hallux disorders, especially hallux rigidus. Taken together our findings suggest the need for careful examination of the proximal FHL and surrounding structures at the entrance into the fibro-osseous tunnel when diagnosing and treating disorders of the great toe and 1st MTP joint.

REFERENCES


AFFILIATED INSTITUTIONS FOR CO-AUTHORS

**George Washington University, Washington, DC